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Louise Brearley Messer ANZSPD Post-graduate Essay Competition

A new class of anticoagulant drugs, the new oral anticoagulants or NOAC's, have begun to play a role in the management of thrombotic diseases. Discuss: (1) Thrombotic disease in children and whether there may be a place for NOAC's in the management of these children, and (2) The dental management of patients on anticoagulants and how this would need to be altered for any children taking NOAC's.

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Abstract

Recently, there is an increase in the incidence of thrombotic events in children requiring treatment or prophylaxis with new oral anticoagulants (NOACs). Currently, there is insufficient evidence on the use of NOACs in children. Nevertheless, several studies related to NOACs are currently underway, hence should provide evidence regarding the dental management of such patients in the near future. Nonetheless, clinicians should have a clear understanding of their mechanism of action, pharmacokinetics, drug interactions etc. Detailed assessment is essential prior to proceeding with NOACs treatment especially in children. It is envisaged that the conclusions from ongoing clinical trials will facilitate development of a pediatric anticoagulation guideline. Until then, it is at the discretion of practitioners to examine, liaise and manage patients in a multi-disciplinary involvement and case specific needs. Any decision to withhold anticoagulant medication should be made in conjunction with the patient's medical practitioner, with consideration of bleeding risk compared to the risk of a thromboembolic event.

Thrombotic disease in children

Recently, there has been a gradual increase in the incidence of thrombotic disease in children (a child is a person under 18 years of age a according to the Australian federal law). The trend comes from a true rise in the incidence which is as a result of improved care, surgical advances, and technology in care of critically ill children parallel with increased detection. Most thrombotic events are secondary complications of severe underlying diseases and the highest risk factor being central venous or arterial lines.

The current knowledge of thrombotic events in children has developed mainly from registries, administrative databases, and retrospective cohort studies. The reported incidence of thromboembolism in children in developed countries ranges from 0.07 to 0.49 per 10,000 children.[1-3] An epidemiology study of venous thromboembolism in children at the Royal Children's Hospital in Melbourne in a 4 year prospective registry found the incidence in children (in hospital admissions) to be eight in 10,000, with 58% being infants and 49% male.[4] The peak of venous thromboembolism in the neonatal period tends to be 20% and in about 50% cases a second peak occurs during adolescence.[4] The relatively higher incidence in neonates as compared to older children may be due to higher haematocrit, and the greater liability of the haemostatic system in neonates due to the generally decreased levels of both coagulation factors and their inhibitors in this age group, except factor VIII (FVIII) and von Willebrand factor (VWF), which are normal or even elevated.[5] In adolescents the incidence equals that of young adults, probably due to the

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THIS ISSUE

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References: 1. Prasad K, et al. J Clin Dent 2018;29 (Spec Iss A):A25–32. 2. Delgado E, et al. J Clin Dent 2018;29 (Spec Iss A):A33–40.

...continued From page 1

hormonal status, the use of contraceptives or pregnancy in young women, obesity and smoking.6 Therefore, the epidemiological background needs to be factored in when assessing the individual risk of thrombotic events in children. Within the paediatric population, higher incidence of thrombotic events are found in children with critical illness, neoplasm, renal disease (nephrotic syndrome), congenital heart disease, inflammatory bowel disease, trauma, sepsis, immobility, total parenteral nutrition, contraceptives or obesity and neonates.[7]

Current anticoagulant treatment

Oral anticoagulant therapy is commonly used for prophylaxis and management of thrombotic disease in children. The administrative use of anticoagulants in children is usually complicated with multiple medical issues and several medications taken by the patient. The most commonly used anticoagulants in children are unfractionated heparin, low molecular heparin, and the vitamin K antagonists (VKA). However, these anticoagulants have several challenges in children (see Table 1).

The mechanism of action of heparins is by endogenous antithrombin which has plasma levels that are physiologically low in neonates or may be decreased in unwell children.[8,9] Unfractionated heparin is frequently used for short-term prophylaxis and in acute illnesses.[7] Conversely, low molecular heparins have more stable, age-dependent, pharmacodynamic properties, and require less regular monitoring than un-fractioned heparin. The longer half-life makes them useful for long-term use, including in the outpatient setting.10 Heparin is delivered parenterally which is useful in sick patients, however long term it is impractical in children.[10] Also, as they are only available in dose vials for adults, a certain amount is initially disposed or diluted which can lead to medication errors, and is tedious and wasteful.[7]

The VKA are currently the only oral anticoagulants available for children. They have a slow onset and offset, are influenced readily by food intake, and have multiple drug interactions.[11] These downfalls are heightened in children who often have dietary problems, multiple medical issues and usually are on multiple drugs with potential to interact with VKA. In neonates, VKA use is exceptionally problematic due to inconstant vitamin K intake through breast milk or formulas. Other challenges with VKA in children include: frequent venepuncture to monitor VKA levels, access to age-appropriate administration forms and dose strengths covering the wide range of doses required, unavailability in a liquid formulation.[11] Therefore, there has been a high demand and innovation for the development of a novel oral anticoagulant that is safer and more effective. Table 2 summarises thrombotic diseases in children and indications of use for anticoagulants.

New oral anticoagulants (NOACs)

The new oral anticoagulants (NOACs) are small molecules, designed to selectively inhibit specific coagulation factors in the coagulation pathway.[12] These NOACs work primarily by two mechanisms, which target specific coagulation factors either in the form of thrombin or activated coagulation factor Xa. The relevant pharmacological properties of the currently approved anticoagulants is summarised in Table 3. Dabigatran directly binds to clotting factor IIa (inhibits thrombin) whereas rivaroxaban, apixaban, edoxaban, and betrixaban inhibit factor Xa, VKA reduces clotting factor production and heparin binds to antithrombin III to induce anticoagulation.[13] This mechanism was novel for an oral anticoagulant and even for most parenteral anticoagulants. Subsequently, the term "new" has been replaced in more recent literature as "direct" however for the purpose of consistency, NOACs will be used throughout this essay.

New oral anticoagulants for children

The potential advantages of NOACs in children have been suggested from pharmacological properties of NOACs and clinical results from adult trials: oral administration, predictable pharmacokinetics, no anti-thrombin dependence, no food interaction, few drug interactions, wider therapeutic window, and possibly no monitoring requirements.[13,14] To date, NOACs have not yet been approved from use in children in Australia. All NOACs have 'Paediatric Investigation Plans' (PIPs) that must be approved by these regulatory bodies and are ongoing.[15] A comprehensive list of the indications targeted by current PIPs for rivaroxaban, dabigatran, apixaban, edoxaban, betrixaban, comprising prophylaxis of venous thromboembolism (VTE), prophylaxis of arterial/cardiac

Table 1. *Comparison of currently used oral anticoagulants in children.*

	Heparin		**Vitmain K antagonist (Warfarin)**
	Unfractionated	**Low molecular weight**	
Advantages	· Fast onset/offset · Titratable · Reversible	· Weight adjusted dosing · Predictable pharmacokinetics/ pharmakodynamics · Reduced monitoring requirements · Relatively quick onset of action (hours)	· Oral route of intake · Suitable for long term use (years) · Quick reversal (vitamin K)
Disadvantages	· Inter and intra individual dose variation · Intravenous access – Requires frequent monitoring · Reduced elimination in renal impairment	· Requires venipuncture · Reduced elimination in renal impairment · Difficult to reverse	· Affected by diet, other drugs and treatment, intercurrent infection · Affected especially in infants with variable vitamin K levels · Slow onset and offset · Unavailable in liquid formulation
Use	· Short term prophylaxis · Acute illness	· Short/moderate term anticoagulation in either inpatient or outpatient setting	· Moderate to long term anticoagulation in compliant patients

Table 2. *Comparison of common anticoagulants and new oral anticoagulants (NOACs).*

	Dabigatran	Rivaroxaban	Apixaban	Edoxaban	Warfarin	Heparin
Mechanism of action	Direct thrombin inhibitor	Factor Xa inhibitor	Factor Xa inhibitor	Factor Xa inhibitor	Reducing clotting factor production	Endogenous antithrombin
Elimination	80% renal	67% renal, 33% faecal	25% renal, 75% faecal	35% renal	Hepatic, primarily CYP2C9	Renal
Bioavailability	6%	80%	50%	50%	80%	20%
Monitoring	Hemoclot thrombin inhibitor assay	Anti-Xa assay	Anti-Xa assay	_	INR	Activated partial thromboplastin time
Reversal	Haemodialysis Infusion of PCC has shown mixed results Administration of recombinant Factor VIIa may be useful	Non-dialysable Infusion of PCC Administration of recombinant Factor VIIa may be useful	Non-dialysable Infusion of PCC Administration of recombinant Factor VIIa may be useful	_	Administration of vitamin K infusion of PCC or FFP if life-threatening haemorrhage	Protamine sulfate
Brand names	Pradaxaâ	Xareltoâ	Eliquisâ	Lixiana		

PCC = Prothrombin complex concentrate. FFP = Fresh frozen plasma.
* assumes no renal impairment

Table 3. *Indications for anticoagulant prophylaxis and new oral anticoagulants treatment in children.*

Prevention of venous thromboembolism	Prevention of cardiac, arterial thromboembolism	Treatment of thromboembolism
· Central venous lines · Peri-operative prophylaxis · Peri-procedural prophylaxis (e.g. cardiac catheterization) · Extracorporal circulation (haemodialysis, extracorporal membrane oxygenation, cardiac bypass surgery)	· Central arterial lines · Mechanical heart valves · Shunts (e.g. Fontan); stents · Dilated cardiomyopathy · Kawasaki syndrome · Ventricular assisted devices	· Venous thrombosis, pulmonary embolism · Cerebral sino-venous thrombosis · Arterial ischemic stroke · Cardiac, arterial thrombosis

thrombosis, and treatment of VTE is listed in Table 3. Furthermore, the details of all current randomised controlled clinical trials on NOACs including their timelines and current status is listed in Table 5.[16,17] A fundamental element is the development of age-appropriate paediatric formulation(s) to ensure reliable and accurate administration to specific age groups of children. This includes studies to establish bioequivalence with the adult formulations. For clinical studies, most PIPs on NOACs perform single-dose pharmacokinetic and pharmacodynamic(PK/PD) studies in children of all age groups for initial dose-finding and safety assessment.[18] Only the dabigatran programme involved a multiple-dose PK/PD study over three days in adolescents and a few children proceeded with single-dose assessments in younger children.19 Phase 1 studies on anticoagulants cannot be performed on healthy children but usually target children with previous thrombosis shortly after the end of a course of anticoagulation.[18] Such studies are ethically challenging as short-term exposure to the drug offers no therapeutic benefit and repeated venepunctures for PK/PD samples are a significant burden for the child. The studies on venous thromboembolism treatment have limited patient numbers between 150 and 274, thereby, not powered to independently demonstrate efficacy or safety in children. The relatively small sample sizes are explained by the feasibility challenges of such trials.[20, 21] The findings from these clinical trials will demonstrate whether extrapolation from adult trials is feasible and the overall data from adult and paediatric studies should provide sufficient evidence to conclude on efficacy and safety of NOACs in children. Thus, we may expect most NOACs eventually to be authorized for children, providing paediatric formulations, and data from clinical trials regarding age-specific dosing, efficacy and safety.

Challenges of paediatric studies with direct oral anticoagulants

The current studies are focusing on rationalisation in paediatric use to ensure they target diverse indications and address the whole spectrum of paediatric indications for anticoagulation. Given the diverse and severe underlying diseases in children suffering from thrombosis, it is difficult to define eligibility criteria for clinical trials that reflect the real-life population of children requiring anticoagulation.[22] Recruitment is further limited by difficulties in obtaining parental consent and also the clinicians' reluctance to randomise. Moreover, adherence to study assessments, particularly if they involve frequent blood sampling for PK studies, or radiographic investigations, can be challenging in small and sick children. It is important to find the right balance between stringent study protocols to optimize scientific validity and study burdens acceptable to children and their families.

Is there a role of NOACs in children?

To date, there is insufficient evidence to recommend the use of NOACs in children. For the near future, the pharmacological properties of the NOACs and the special characteristics of children requiring anticoagulation, the NOACs have the potential to be of particular benefit for children. If these advantages outweigh the results of the current studies, the NOACs will likely be used widely in children. Even if shown to be equal to conventional anticoagulants with regards to safety and efficacy, the increased convenience of NOACs will likely favour their use. Moreover, in real-life practice increased convenience may translate into improved compliance with the potential for increasing safety and efficacy. As oral drugs, the NOACs will be most beneficial for long-term anticoagulation. Further studies investigating the role of NOACs for use as prophylaxis or initial treatment in children are required.

Use in acutely ill children, particularly infants and neonates, will require that administration through gastric feeding tubes is validated, and that enteral absorption is reliable. Given the differences between NOACs in oral bioavailability, not all drugs may be equally applicable for acutely ill or very young children. Differences between NOACs in the ratio of renal versus hepatic clearance may also render certain drugs more or less applicable for children with renal insufficiency or hepatobiliary disease.

The current state of approved NOACs have paediatric developments ongoing or planned, some of which are substantially progressed into phase 3 studies. Formulation of NOACs to suit the specific paediatric population have been developed and are currently being trialled. A fundamental factor for use in children is to produce clear age-appropriate dosing guide for each age group. Unfortunately, the PK/PD studies of current PIPs have very small numbers of infants and neonates, and premature infants are usually excluded.[7,20] Conversely, pharmacological and haemostatic differences are most pronounced in the youngest age groups, with a potential for differences in PK and/or PD.[7] Similarly, the phase 3 studies are struggling to recruit younger children. Nevertheless, the critical situations where the need for therapeutic drug monitoring in children needs to be elucidated. It is possible that in very young children (infants, neonates, premature), acutely sick children, and children with relevant comorbidities, monitoring drug levels or activity may be required to establish the initial dose, or for dose adjustments during changing clinical situations. In addition, monitoring may be necessary for acute surgery, bleeding or thrombotic complications, and possibly, to assess compliance.

The results of ongoing clinical studies in children still have to demonstrate the positive benefit risk balance for NOACs in all targeted paediatric indications and age-groups. This accounts particularly for indications that have not been explored in adults such as prevention of catheter-related VTE, or anticoagulation for congenital heart disease, such as Fontan surgery. The risk and type of bleeding may be different in the paediatric population. For example, epistaxis is a more common bleeding complaint in preschool and school-age children, as compared to older patients. Menstruating females will also make up a substantial percentage of paediatric trials and special attention will need to be paid to reproductive bleeding in this sub-population. Although there is limited evidence, in adult populations, studies have shown rivaroxaban in particular may have a higher rate of heavy menstrual bleeding as compared to traditional therapies.[23] It will not be possible to clarify all these questions from pre-authorization studies in children. Thus post-authorization studies will be required to generate more data on long-term safety and efficacy, age group specificity (especially in neonates), and other paediatric special disease populations.[22]

In light of the current literature, NOACs should not be used off-label in children and adolescents. The preliminary results still put children at risk as there are still unknown, unanswered questions and insufficient information available on dosing, safety and efficacy. Additionally, off-label use endangers recruitment of children into the ongoing PK/PD and comparative studies through loss of equlibirum.[22] The regulatory requirements for paediatric drug development provide unique chances to obtain systematic data for NOACs in children. On the other hand, there are significant challenges to performing valid and informative drug studies in children. Therefore, all efforts should currently go into treating children with NOACs within the ongoing studies. However, in the near future, given expected finish times of ongoing PIPs, paediatric use of certain NOACs may be approved. The field of paediatric medicine should be encouraged to promote participation of children and adolescents into the multiple ongoing studies of NOACs where feasible, to allow for stronger evidence to support management strategies in paediatric anticoagulation.

Dental management of patients on anticoagulants

Due to the pharmacological advantages there is a trend in decreased use of VKAs and increase in the number of patients on NOACs in the adult population and potential for use in children. Lack of defined management guidelines for such patients and paucity of clinical data in this field, forces the dental professional to rely on pharmacological data for these drugs along with their experiences. Few evidence based guidelines have been published and require updates.[24-27] A detailed medical history is required including use of prescription medications (e.g. antiplatelet therapy) and complementary medicines including fish oil, gingko and glucosamine, which can also increase bleeding.[28]

For dental management of patients taking NOACs, the potential risk of bleeding should be weighed against the risk of stroke or a potential thromboembolic event. There are several factors that should be considered prior to treatment in order to determine whether a patient should cease their NOAC, and if so, how long before treatment this should begin. The clinician needs to identify the dental procedure's associated risk of bleeding (see Table 6). If the dental treatment is known to be an unlikely cause of bleeding, then there may not be an indication to discontinue NOACs prior surgery.[29, 30] However, for

Table 4. *Indications targeted by current Paediatric Investigation Plans for Direct Oral Anticoagulants.*

	Dabigatran	Rivaroxaban	Apixaban	Edoxaban	Betrixaban
Prevention of Venous thromboembolism			Acute leukemia/ lymphoma, asparaginase treatment, with central venous catheter (apixaban versus placebo)		1. Medical illness or surgery (betrixaban single arm) 2. Neonates/ preterms with umbilical catheter (betrixaban single arm)
Prevention of Cardiac, arterial thromboembolism		Post Fontan surgery 2 (rivaroxaban versus aspirin)	Various cardiac diseases (apixaban versus SOC)	Various cardiac diseases (edoxaban versus SOC)	
Treatment	1. Acute VTE (dabigatratran vs SOC) 2. Extended secondary prevention (dabigatran single arm)	Acute VTE (rivaroxaban vs SOC)	Acute VTE (apixaban versus SOC)	Acute VTE (edoxaban versus SOC)	

Table 5. *Current Paediatric Investigation Plans on new oral anticoagulants*

Randomised controlled trial	Age	Phase	Number of patients	Status	Reference	Expected completion
Dabigatran						
Treatment of VTE	0-18	2b/3	180	R	Halton et al.[19, 21]	2018-Q2
Rivaroxaban						
Treatment of VTE	6-18y	2	63	C	Attard et al.[45,46]	2017-Q4
	0-18y	3	270	R		2019-Q3
Prevention of cardiac/arterial TE	2-8yo	3	100	R		2022-Q1
Apixaban						
Prevention of VTE	1-18y	3	500	R	Yetman et al.[47]	2019-Q4
Prevention of cardiac/arterial TE	0-18y	2	150	R		2020-Q2
Treatment of VTE	0-18y	3	150	R		2020-Q4
Edoxaban						
Treatment of VTE	0-18y	3	274	R	Yetman et al.[47]	2020-Q4
Prevention of cardiac/arterial TE	0-18y	3	150	NR		2021-Q1

Abbreviations:
PIP, Paediatric Investigation Plan
VTE, venous thromboembolism; TE, thromboembolism
C, studies completed; P, results published; O, ongoing; R, recruiting; NR, not yet recruiting; Q1/2/3/4, quarter of that year;
Information based on published Paediatric Investigation Plans and study details and study status reported on www.clinicaltrials.gov

Table 6. *Bleeding risks for dental procedures.*

Low risk	Moderate risk	High risk
Local anaesthesia	Simple extractions (less than 3 teeth, restricted wound size)	Complex extractions, adjacent extractions that will cause a large wound or more than 3 extractions at once
Periodontal examination	Subgingival periodontal debridement	Procedures involving flap raising
Supragingival removal of plaque, calculus and stain	Incision and drainage of intra-oral swelling	Periodontal, preprosthetic, periradicular surgery
Direct or indirect restorations with supragingival margins	Direct or indirect restorations with subgingival margins	Dental implant surgery
Endodontics—orthograde		Gingival recontouring
Impressions and other prosthetics procedures		Crown lengthening
Fitting and adjustment of orthodontic appliances		Biopsies

procedures that are associated with low and high levels of bleeding risks, NOAC discontinuation should be considered.[20, 31]

Monitoring of anticoagulants

The NOAC agents were marketed to provide predictable and consistent anticoagulant therapy, without monitoring.[32,33] However, monitoring of anticoagulant efficacy may be important assessing bleeding risk in an emergency setting.[33] Traditional laboratory tests are difficult to interpret with NOAC therapy, since agents such as dabigatran and rivaroxaban variably affect coagulation markers.[34] To overcome these issues, some clinical laboratories use specific tests, in conjunction with a haematologist however, it is important to note that these coagulation tests are highly specialized and may not be available in every laboratory.[24, 34]

Reversal agents

The reversal of anticoagulant therapy may be required, for example in case of major haemorrhage. Such indications are rare in outpatient dental settings, but awareness of reversal agents may be beneficial. In situations involving post-operative bleeding, local measures should be employed. Reversal agents should only be considered, in conjunction with the patient's medical practitioner, if bleeding continues despite conservative haemostatic management.[33] Recombinant factor VII has shown potential promise in animal and in vitro studies in reversing both rivaroxaban and dabigatran, but has been inconsistent so far and requires further investigation.[35] Currently, discontinuation of the NOAC may be sufficient to control mild bleeding in patients with normal renal function, since the drugs have short half-lives[35,36] and has been successfully demonstrated in a case report.[37]

Development of reversal agents for NOACs for children

Given the potent anticoagulant effects of NOACs, there is a need for reversal agents to prevent bleeding in case of emergency surgical procedures and to treat major haemorrhages. Idarucizumab, a reversal agent specific for dabigatran, has been developed and is approved for adults.[38] A PIP for idarucizumab is ongoing, consisting of a single dose study of idarucizumab used as rescue medication in children, and a registry of paediatric patients treated in practice with idarucizumab.[39,40] Another agent, ciraparantag, designed to reverse the effect of a wide range of anticoagulants (UFH, LMWH, other parenteral and oral factor Xa inhibitors, dabigatran, argatroban) is in early clinical development and therefore there is no established PIP.[41]

Management guidelines for children on NOACs

The recent introduction of the NOACs means that there have been limited randomized control trials and only generalized management protocols have been established.[27, 42] Therefore, it is advisable that caution is observed with patients taking NOACs, and that randomized clinical trials be used to establish formal, dental-specific management guidelines, especially in children. In the meantime, a conservative approach should be adopted by evaluating both the bleeding risk of the proposed treatment and the thrombotic risk of modifying the anticoagulant regimen, which should only be considered in collaboration with the patient's medical practitioner. Liaising with an appropriately qualified and experienced colleague is recommended. Conservative haemostatic management include local measures such as mechanical pressure, haemostatic agents (such as Gelfoam™ or Surgicel™), suturing and tranexamic acid mouthwash.[24] Procedures which are associated with an increased risk of bleeding may require cessation of anticoagulant medications before surgery.[43] The time period of cessation of NOAC therapy is dependent on each patient's renal function, specific anticoagulant and dental procedure involved. If bleeding continues and does not respond to local measures, or if there is spontaneous bleeding, urgent medical attention is required, with consideration of referral to a tertiary centre for assistance in management.[24]

Currently, there are no standard guidelines on the management of major haemorrhage. Therefore, a specialist service (e.g. haematology, critical care, or internal medicine) should be consulted,

with a case-by-case approach.[20] Liaising with the medical practitioner and having a detailed knowledge of the patient's medical condition, planned dental procedure and bleeding risk are imperative in patient management.

Conclusion

As the use of the NOACs increases, it is important that dental practitioners are aware of these drugs, and their mechanisms of action. Pending future research clinical guidelines for NOACs, including use in dental practice should be readily more available (similar to those currently available for warfarin). Any decision to withhold anticoagulant medication should be made in conjunction with the patient's medical practitioner, with consideration of bleeding risk compared to the risk of a thromboembolic event.

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Federal President's Report

Dr Sue Taji

I am writing these lines whilst aboard a Qantas flight back to Brisbane, reflecting upon a wonderful few days at ANZSPD's 2019 RK Hall event in Perth.

Full credit is due to the local organising committee of the WA branch, who have worked tirelessly to organise the event. The international speakers and those from within our region provided thought provoking, timely and clinically orientated presentations that provided relevant updates on various topics within paediatric dentistry and paediatric health overall.

Progress in our field is achieved through research, education and continuing professional development. One of ANZSPD's aims within the society is to continuously advocate for the furthering of education within the field of paediatric dentistry. Beyond great regional meetings, such as the recent 2019 RK Hall event in Perth as well as ANZSPD's biennial events, there is arguably no better way but to consider holding the international congress on paediatric dentistry here in the Australia and New Zealand region.

Within the ANZ region, the International Association of Paediatric Dentistry Congress (IAPD) was last held in Sydney close to two decades ago in 2005. Prior to that, Melbourne hosted the IAPD congress forty or so years ago in 1983. These are the only two occasions where an international congress specific to the field of paediatric dentistry has been held in the ANZ region. The rules of the IAPD state that a minimum of twenty years need to elapse prior to any region holding another international congress. Federal councillors and the executive committee have voted for ANZSPD to pursue the idea of holding this international congress in our region in the not so distant future and for Melbourne to be the preferred host city.

Plans have been set in motion to place a bid for the 2025 IAPD congress, to bring this International congress to our region and to have it held in Melbourne. Such an event will bring more great minds within the field of Paediatric Dentistry from far and wide and give colleagues the opportunity to attend a world class congress specific to the field in our region. Such an international event within our field will combine a remarkable line-up of leading international experts who collectively are very much at the forefront of the field. Whilst 2025 sounds like a long way away, realistically it's only five and a half years away and if ANZSPD's bid is successful, there will be avenues down the track where interested members can get involved in the organisation of this major event and I would like to encourage any interested member in due course to get in touch with your Federal Councillor if you are interested in being involved.

Another topic that has received plenty of time and thought by many has been the revision and modernisation of the ANZSPD's Federal Constitution to bring this important document in line with current best practice. The Federal Councillors and Executive team have all had their input in this process. However, I particularly wish to acknowledge and make special mention of Dr John Sheahan and Dr Karen Kan for their tremendous efforts and enthusiasm in this task. At the AGM that was recently held at the RK Hall event in Perth, on behalf of the Federal councillors, the Federal Executive and the ANZSPD membership, I took the opportunity to thank Drs John Sheahan and Karen Kan for their efforts and do so again now.

At ANZSPD's AGM, the membership voted in favour of adopting the updated Federal Constitution. With this positive step for our society now set in stone and with the new Federal Constitution now in place, it is worthwhile for all branches to consider checking and reviewing their own branch constitution such that these guiding documents may work in tandem with the Federal Constitution for many years to come.

Special mention is also due for the excellent work Dr Steven Kazoullis has contributed to the ANZSPD, not only in his ongoing editorship of the quarterly Synopsis magazine, but also through undertaking the tedious and time consuming task of digitising many of ANZSPD's archival records as part of our drive to modernise aspects to ensure better future efficiency.

Finally, through our collaborations with the International Association of Dental Traumatology (IADT), we are now pleased to be offering all our members free access to this very valuable online resource, the Dental Trauma Guide. All members should have received an email with directions as how to utilise this valuable resource and hopefully this has made it through to you.

Kind regards,

Dr Sue S. Taji, *ANZSPD President*

ANZSPD (NSW) **Branch Report** 2018

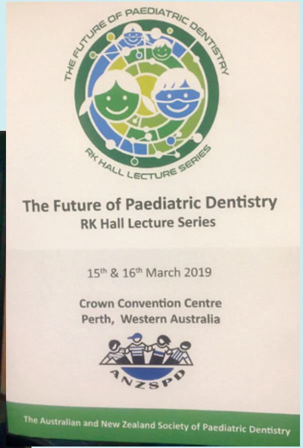
President: Dr Prashanth Dhanpal
Secretary: Dr Jason Michael
Treasurer: Dr Maansi Juneja
Federal Councillor: Dr Soni Stephen
Committee members: Dr Naveen Loganathan (Immediate Past President)
Dr Eduardo Alcaino
Dr Sherene Alexander

The ANZSPD NSW branch had a very busy and memorable 2018. We hosted three dinner meetings and a full day scientific meeting. We have been working hard to increase membership and attendance to our events. Our professional development commenced with Dr Stephen Harlamb (endodontist) discussing the controversial topic exploring the endodontic management of incompletely developed permanent teeth in March 2018. He presented a balance view exploring the options of regenerative endodontics and traditional apexification modalities. Dr David Elliott (anaesthetist) presented the topic 'General anaesthesia for paediatric dentistry – what to say when parents ask "is it safe"? in June 2018. Dr Elliott provided an informative and entertaining lecture that involved considerable audience interaction. Our final dinner meeting for the year was held in August 2018 and was presented by Dr Nour Tarraf (orthodontist). He has been an early adopter of new technology that is disrupting the orthodontic world including 3D scanning and milling, lingual orthodontics, advanced temporary anchorage devices and the application of robotics to orthodontics. More importantly he provided practical insight into how this helps patients, families and practitioners.

Our busy year culminated in our full day scientific meeting in October 2018. Prof Monty Duggal, our keynote international speaker, focused on management of dental trauma. He was supported by local and interstate speakers. Dr Lydia Lim (OMFS) shared wisdom she continues to accumulate with her time spent at the Children's Hospital at Westmead. Dr Jamie Lucas shared an update on innovations in paediatric restorative dentistry and number of impressive cases. Dr Craig Brown shared his thoughts and guidance with regards to dento-legal issues for those providing dental treatment to minors. Prof Ali Darendeliler provided an informative lecture discussing orthodontic management considerations in the mixed dentition.

We are looking forward to a busy 2019 and are working hard to increase our membership base by providing high quality continuing education.

RK Hall
Lecture Series 2019



The 2019 ANZSPD RK Hall Lecture Series proudly sponsored by Colgate was held on 15th-16th March, 2019, at Crown Perth.

The program held a wide appeal, with over 100 delegates including an even spread of therapists, dentists, postgraduate students and paediatric dentists. We were delighted to welcome half of the delegates from interstate or New Zealand, including the lecture series' namesake and special guest, A/Prof Roger Hall and his wife, Vera.

The 2019 RK Hall Visiting Lecturer was A/Prof Peter Day from Leeds, UK. Peter is passionate about evidence based paediatric dentistry and shared with us his current projects relating to the management of dental caries in the primary dentition, developing a core outcome set for traumatic dental injuries and supporting dental teams to have effective behaviour change conversations with parents of young children. Peter captivated the audience with his enthusiasm and knowledge and gave an insight into a UK perspective of paediatric dentistry.
Peter was supported by a fabulous range of local speakers, including Paediatric Dentists, Orthodontist, Prosthodontist, Oral and Maxillofacial Radiologists, Dental Technician, Paediatric Endocrinologist, Paediatric Anaesthetist, Professor of Autism Research, Sports Physiotherapist and a Motivational Speaker.

Complimenting the scientific program was the social calendar. Day one concluded with a sundowner event including cocktails and canapes, an acoustic guitarist and a photobooth. The Gala Dinner was held on the Saturday evening following the conclusion of the meeting at Crown Perth. The quality of company, entertainment (a string quartet playing classical and contemporary covers), food and wine was second to none and this event provided an excellent networking and socialising opportunity. Overall, the 2019 ANZSPD RK Hall Lecture Series was a great success due to the quality of speakers, delegates and venue and we look forward to welcoming everyone back to Perth for the ANZSPD Biennial Meeting in 2021/22 (date to be confirmed).

Louise Brearley Messer ANZSPD Undergraduate Essay Competition

Dental caries in children- infectious disease or parental lifestyle choice? Discuss your views based on current knowledge of aetiology and epidemiology of the disease.

Mr Kumidika Gunaratne
Year 4, School of Dentistry, University of Sydney

Dental caries is defined as the bacterially-produced acid-demineralization of enamel or dentine resulting in a net loss of tooth structure. The pathology involves a bacterial biofilm, a substrate of fermentable carbohydrates and acidic bacterial by-products destroying enamel and dentine. The disease cannot be attributed to an individual host or bacterial factor. Rather an imbalance of oral hygiene, diet, fluoride exposure and other environmental components leads to the progression of the disease. In children, especially those under the age of 6, hygiene and diet are dictated by their parents' lifestyle. The focus of this discussion will be on, but not limited to, risk factors for early childhood caries (ECC) and severe early childhood caries (S-ECC) as children in this age bracket are most reliant on their parents. The evidence to be discussed will show how parental lifestyle factors are the most important determinants of childhood caries, despite the possible contribution of intrinsic host factors. These lifestyle factors include oral hygiene, socioeconomic status (SES), psychosocial factors and feeding patterns. It is important to identify the modifiable risk factors for dental caries in children but also recognise the socioeconomic limitations faced by high-risk populations.

Caries, the infectious disease?

Infectious diseases are "caused by pathogenic microorganisms, such as bacteria viruses, parasites or fungi; the diseases can be spread directly or indirectly, from one person to another".[1] Bacteria such as Streptococci Mutans can be transmitted (vertically) from mother to child,[2] but also (horizontally) from child to child in a day care.[3] However, these bacteria also exist passively in all individuals. Children are not born with the caries disease! Caries only exists via a partnership between acidogenic bacteria and poor lifestyle factors such as a cariogenic diet, poor oral hygiene and low fluoride exposure. With this in consideration, caries can be viewed as a lifestyle-modified bacterial disease which for children, is modified by parental lifestyle choices. If left untreated, dental caries can be fatal as seen in the 2007 death of a 12-year-old boy in the USA because of spread of an abscess to the brain.4 Although childhood caries is biologically understood, it is still prevalent because it is driven by parental and community factors which are hard to modify. Though caries is not communicable, the continuity between parent and child's oral hygiene is akin to the spread of an infectious disease which is why parental lifestyle is pivotal.

Epidemiology of childhood caries

Early childhood caries (ECC) is diagnosed by the presence of 1 or more decayed, missing or filled teeth in primary teeth of a child under the age of 71 months (under 6 years).[5] Severe early childhood caries (S-ECC) is defined as either smooth surface caries in children under 3, or 1 or more cavitated, missing or filled smooth surfaces in primary maxillary anterior teeth or a DMFT score of 4 (age 3), 5 (age 4) or 6 (age 5). This disease is the most common chronic childhood disease.6 Although Australian childhood caries declined up till the 21st century, there has been a gradual increase ever since and now more than half of Australian 6-year-olds possess decay on their primary teeth.7 Internationally, the incidence of ECC is alarmingly high. More than a quarter of preschoolers in America are afflicted with ECC.8 A 32% ECC rate in Sri Lanka infants was linked with culturally-sensitive risk factors like milk tea, socioeconomic factors such as maternal unemployment and health care factors such as access to dental care whilst pregnant.[9] This reinforces childhood caries as a multifactorial disease. A 1991 study found that Finnish children had maxillary incisors that were 3 times less affected by caries than children in Tanzania,[10] reflecting a cultural and perhaps socio-economic disparity in childhood caries.

Child-level factors

Bacterial load in a child's mouth

There is evidence of a link between increased bacterial load and ECC, however we must interpret these results carefully. An increased level of S.mutans in the saliva and plaque of pre-school children in Beijing has been associated with ECC.11 Elevated S. Mutans, S. Sobrinus and Bifidobacteria numbers have also been found to coincide with cariogenic food[12] and bottle feeding.13 We can see then that bacterial presence is essential for childhood caries, but its impact on teeth is likely regulated by environmental factors. Furthermore, studies on the risk factors for ECC often produce multiple statistically significant findings, not isolated ones. For example, the same 1998 study found a correlation between S-ECC and three dietary variables; sipping juice between meals, snacking frequency and lack of protective foods such as cheese. Considering bacterial load as a high priority risk factor is not helpful in caries prevention strategy because antibiotic treatment is ineffective and may be unpleasant for the child.[14] Colinisation of these bacteria is assumed to be inevitable and increases with the eruption of teeth.[15] Instead, the modification of parental and child lifestyle factors is more appropriate for preventing caries. Treatment of these risk factors will incidentally reduce the colinisation of cariogenic bacteria, however quantitative benchmarks are

not necessary for the establishment of a healthy oral environment.

Our mouth undergoes cycles of demineralization and remineralisation every day. Dental caries can be halted, but there is always the possibility of relapse with inadequate oral hygiene and violation of risk factors. Rather than being caries-free, the idea of being "caries- inactive" is a more realistic goal.[16] Therefore caries is not cured but managed by professionals and by parental lifestyle choices.

Tooth morphology

Morphological features of primary teeth render them more vulnerable to tooth decay than adult teeth. Primary teeth have thinner enamel and larger pulp chambers. The mesial pulp horns are also high so there is a greater chance of pulpal involvement. Often there is not enough time for secondary dentine to be laid down in response to pulpal injury. The cervical ridges are bulbous and the Cemento-enamel junction is constricted in molars- both bear restorative implications. Broad interproximal contacts can mask decay and have restorative implications in cavity design.[17] The roots of the primary teeth flare out over the adult follicle underneath, and caries or premature tooth loss may affect the adult successor.[18]

Salivary Hypofunction

Salivation is an autonomic system that is advantageous for caries prevention. Salivary hypofunction due to gland abnormalities, medications or radiotherapy leave a child vulnerable to rampant caries if not managed properly.[19] This is due to the multitude of protective functions saliva normally serves in the oral cavity e.g. lubrication, agglutination for selective adhesion of microflora, remineralisation of teeth with calcium and phosphate, antimicrobial properties, clearance of debris and buffering capacity against acidic assault. Saliva also changes properties between rest and chewing, and flow rate is even tailored to the relative acidity of the food being chewed.

Consequently, children with salivary impairment have a greater experience with ECC than those without. Children who mouth-breath may also share a similar experience. The intrinsic vulnerability of a child with hyposalivation makes caries prevention much more difficult, but it should still be treated as a preventable disease where informed parental decision making is pivotal.

Enamel hypoplasia

Enamel hypoplasia renders teeth more susceptible to decay and is a perilous risk factor for ECC.[20] Defects during the secretory amelogenesis stage of tooth formation lead to poor mineralisation of enamel and an eventual quantitative defect in the erupted tooth (thinner enamel), otherwise known as enamel hypoplasia. This enamel is more vulnerable to bacterial-acidic dissolution. An American study showed that children with enamel hypoplasia carried a 10 times greater incidence of decay after statistical adjustment for bacterial count, age and ethnicity.[21] The caries risk in infancy extends into late primary school.[22] Hypoplastic enamel defects also correlate to low birthweight. Longitudinal studies have found that babies with Very Low Birth Weight (<1500g) show more enamel opacities, which later correlate to increased prevalence of caries in teenage years.[23] The enamel defects may arise from impaired pre-natal development of primary tooth buds.[24] In medically compromised children, caries prevention is especially sensitive to parental lifestyle factors.

Water Fluoridation

Water fluoridation plays an important role in reducing ECC. Where toothpaste is lacking, drinking water provides a supplementary source of fluoride to primary teeth. Ideally, children are exposed to fluoridated water from birth. However, access to fluoridated water is not always available, especially in rural populations. Recent research in over 20,000 children of ages 5-14 has shown that those who missed out on fluoridated water for at least 6 months during their childhood carried a greater risk of developing caries in primary and adult teeth, even after adjustment for socioeconomic status.[25]

Parental lifestyle factors in ECC

Early vertical transmission of strep mutans bacteria is associated with an increased risk of ECC.[2] This can occur through pre-mastication, spoon sharing or kissing on the lips. Vertical transmission can reflect the SES of parents.[26] Victorian data suggests that the disparity in oral health between SES groups is already apparent by the time a child starts school.[27] The same is true when comparing indigenous vs non-indigenous children.[28] The impact of SES is so profound because SES modifies risk factors such as oral health literacy, healthcare access, psychosocial behaviour and nutrition.

Socioeconomic Status and Diet

Fermentable carbohydrates provide a substrate for acidogenic bacteria to initiate demineralization. Sucrose causes bacteria to produce the strongest cariogenic by-product; lactic acid. High-frequency, high-sucrose food intake is a behaviour that encourages the disease progression of dental caries in children. The rate of ECC childhood caries is rising in low SES populations despite our better understanding of its risk factors and preventative strategies.[29] This is may partly be because parents do not have access to grocery stores or cannot afford fresh food.[30] Often caloric energy requirements are met with high sugar foods without protein or other valuable nutrients. Another implication of high-caloric diets is obesity. Obesity is linked to health catastrophes such as myocardial infarction and diabetes mellitus but has also been linked with enamel hypoplasia,[21] which elevates caries risk. Malnourishment can also lead to enamel hypoplasia and poor immunological resilience.[24]

Snacks provided from other care takers like grandparents or neighbours also contribute towards caries risk in children.[31] Gas or electricity restriction limits preparation of nutritious food and may promote high caloric fast-food.[32] The result is a greater rate of ECC in low SES families.

School meals

Children at primary school rely on their parents for lunch and recess. When packing food, Perth parents found it difficult to meet all the ideal criteria of appearance, taste, convenience and nutrition.[33] High-caloric snacks such as muesli bars, juice and sweetened yoghurt are frequent constituents of recess and lunch for children, particularly those from low SES.[34] A 2014 Irish study on primary school children found that food consumed at school was higher in sugar content compared to at home.[35] High-caloric snacking provides substrate for acidogenic bacteria. Parents may choose these conveniently-packaged snacks due to time constraints and the necessity to prepare meals for more than one child. School meals may reflect geographical trends, as Australian preschoolers in rural day cares consume more vegetable servings.[36]

School barriers

Interviews report that playground "show and tell" at lunch time exerts peer-pressure and subsequently parental-pressure to buy popular foods. [33] The same study also reported that anecdotal advice from other parents influenced shopping decisions.

Though there are guidelines in place for school canteens, a low proportion of them follow the recommended proportion of healthy, "occasional" and discouraged foods available for purchase.[37] Furthermore, the prices of healthier foods in canteens are often more expensive[38]. More initiatives like the Fresh Kids program in Victoria need to be established in schools to promote nutrition from home rather than reliance on canteen food.[39]

A child's dexterity or wobbly teeth can limit healthy eating. For example, peeling fruit requires patience and sometimes assistance. Another barrier to school nutrition is the lack of refrigeration which may spoil food. Within a few hours of leaving home, a cooled lunchbox can heat to room temperature.[40]

A child's priority at lunch and recess is to play, which means eating time is a deduction of play-time. As a result, they may not finish meals or ask for smaller portions and conveniently packaged sugary snacks to maximise play-time.

Breast feeding and night-time bottle feeding

Breastfeeding is considered the preferred option to bottle feeding in infants. Exclusively breast-fed children may have lower caries-risk than bottle-fed children, however some circumstances such as maternal HIV/AIDs prevent breastfeeding so bottled milk is a necessity[41]. Prolonged breast feeding beyond the age of one is more common in low SES families however some argue this time extension does not impact caries experience[42]. An association exists between night-time bottle feeding and S-ECC.[43] The choice to bottle-feed at night time exposes the child's oral environment to fermentable carbohydrates for extended periods of time, keeping its pH low and promoting demineralization of enamel. To add to this detriment, oral hygiene is often skipped afterwards for the sake of not disrupting the child's sleep. Night-time bottle feeding coincides with the period of slowest salivary flow (sleep) which limits remineralisation and the clearance of residue. On-demand breast-feeding is also harmful and should not be overlooked as a risk factor in addition to allowing children to snack on soda and juice between meals.[44]

Oral hygiene habits

Children under the age of 6 may need manual assistance when performing oral hygiene and it is advisable to always supervise brushing until the age of 8-10.[16] Neglecting these duties may reflect a lack of knowledge or personal beliefs and places infants at high caries-risk. If good oral hygiene is not a pre-natal habit, then it is unlikely to develop in children of that household.[45] Brushing for children poses challenges – as toddlers become more autonomous with speech and movement, non-compliance around brushing time can develop from ages 1-3.[16] Just like low birth weight, maternal smoking has been associated with an increased rate of ECC.[46] Smoking during pregnancy may predict a pattern of neglectful behaviour postnatally and oral hygiene subsequently suffers. Other health issues arising from pre-natal smoking may require the use of sugar-containing or immunosuppressive medications which increase the child's caries risk.

Fatalism

Psychosocial elements also play a role in ECC and contribute to a fatalistic belief about tooth decay. This becomes apparent when families living in the same neighbourhood can have different oral health statuses.[30] Fatalistic beliefs lead to the neglection of oral hygiene duties in both mother and child. These beliefs are potentiated by maternal fatigue and depression.[47] The majority of participants in this study believed caries in their children was inevitable and lacked confidence in their child's brushing.

Education

The recommended time for a first visit is within the 1st year post-natal or within 6 months of the first tooth erupting.5 Delayed visits to the dentists reflect financial constraints, but also educational gaps. For mothers without any schooling, oral health literacy is poor and can cause increased rates of ECC whereas caregivers who finished high school are more likely to seek dental care.[48] A study in a Tanzanian village found that regular dietary and hygiene instruction from healthcare workers was a great tool for ECC prevention.[49] Similar success was found in a Brazilian intervention of low-income households; nutritional advice given to mothers in the child's first year of life significantly reduced the rate of caries by the time children were 4 years old.[50] They found that mothers were compliant with some nutritional advice such as exclusive breastfeeding, delayed introduction of high-sugar foods and taking intervals between meals. However, advice about night-time bottle feeding and introducing fruits and vegetables into the child's diet was ignored. Changing these behaviours might be more difficult due to the psychological aspect of soothing a crying child. Given the long-term instability of educational oral health interventions, pre-natal or intervention within the first year is crucial in forming lasting habits. Delayed intervention reduces the prognosis of establishing

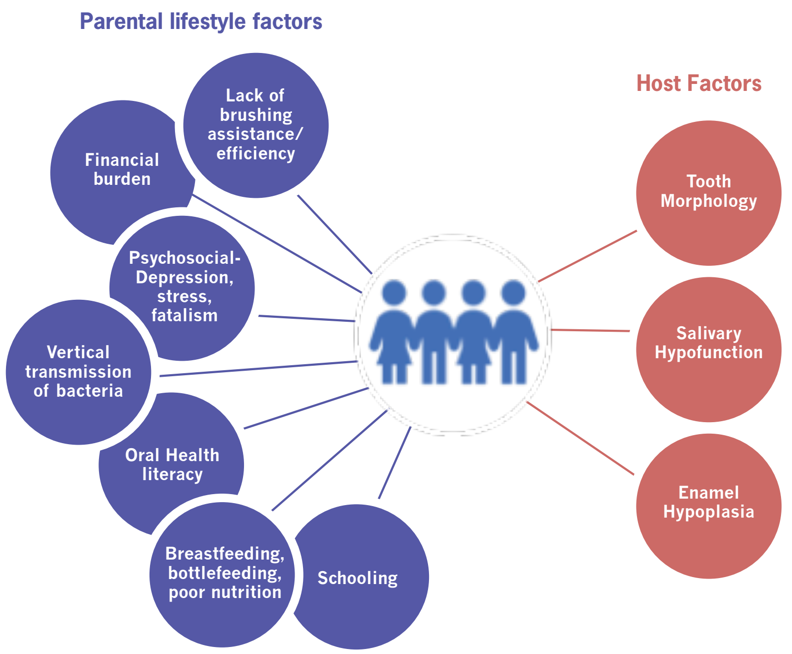


Figure 1. *A diagram illustrating the intrinsic (Child-level) and parental lifestyle influences on childhood caries.*

long-term practices because it requires dismantling of existing habits.

Conclusion

Childhood caries is becoming increasingly prevalent and can cause rapid decay of morphologically-vulnerable deciduous teeth. The prevalence and disparity of ECC is driven by parental lifestyle risk factors such as low SES, home and school nutrition, oral hygiene habits and beliefs, psychosocial behaviours and oral health literacy. They are not totally independent and sadly potentiate each other's effects. In special-risk groups such as children with enamel defects, low birth weight and hyposalivation, poor parental lifestyle factors are even more devastating for a child's oral health.

Habits like pre-chewing food for infants and lip-kissing no doubt contribute towards vertical transmission of bacteria but are unlikely to exclusively drive ECC. The overwhelming trend suggests that parental lifestyle behaviours are causing the burden of caries in children to increase in prevalence in high-risk populations (see Figure 1 on previous page). These parental behaviours are a product of their socioeconomic context and can specifically be attributed to poor oral health literacy, financial restraints, mental health, oral hygiene habits, nutritional patterns and access to healthcare. Management of childhood caries should be sensitive to the child's specific attributes and the role of the caregiver. This is how we can gain a better understanding of their decision-making and how to modify it. By doing this we can tailor oral health intervention strategies to each parent and child. The parental lifestyle risk factors serve as an important reminder that paediatric dentistry should follow a holistic approach to treatment of childhood caries.

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Pre-eruptive intracoronal resorption: Literature review and report of a case assessed with cone beam computed tomography

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Key words: pre-eruptive intracoronal resorption, occult caries, cone beam computed tomography, preventative dentistry

Abstract

Pre-eruptive intracoronal resorption (PEIR) is relatively common, yet poorly understood entity; which over the years has caused confusion in diagnosis and management for the general dental practitioner. This report reviews the literature and presents for the first time, a case of extensive PEIR lesion assessed using cone beam computed tomography. The three dimensional imagining from this radiographic technique aids in accurately determining the pulpal extent and the location of the lesion for better restorative, endodontic and surgical management.

Literature Review

Prevalence

Pre-eruptive intracoronal resorptive (PEIR) lesions are seen on routine dental radiographs as radiolucent defects in dentine of an unerupted tooth. They are often found adjacent to the amelodentinal junction of the crown with no apparent effect to the enamel.[1-3]

PEIR lesions have been reported in the literature over the last 70 years predominantly in case reports. Previous authors have reviewed published case reports outlining over 80 affected teeth.[2,4,5] Since 2012, a further four case reports have been published outlining the management of PEIR lesions associated with single mandibular molar teeth,[6-8] and a mandibular premolar tooth[9] (Table 1).

To determine the prevalence of PEIR lesions in permanent teeth, five studies have analysed bite-wing and panoramic radiographs retrospectively.[4,10-13] The studies have shown that the subject prevalence ranges between 1.5% and 27.3% and tooth prevalence between 0.5% and 2.1% depending on population studied and type of radiograph used. The most common teeth affected are molar and premolar teeth although other teeth have also been reported including incisors and a supernumerary tooth.[4,14,15]

All of the studies have found no gender predilection, no association with medical conditions, and no association with use of fluoride supplements or with drinking of fluoridated water.[4, 10-13] Furthermore, the PEIR lesions are most often found on the occlusal aspect of the crown, adjacent to the amelo-dental junction with a smaller proportion found in the mesial or distal aspects of the crown. At the time of detection, most of the defects are less than one-third of the thickness of dentine.[10, 11]

Majority of cases involve a single tooth while ectopically positioned teeth are involved in a significant proportion of PEIR cases (14%).[4,11]

The prevalence of PEIR in primary teeth

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Table 1. *PEIR case reports published since 2012*

Author, Date	Patient Age, Gender, Tooth affected	Management
Couniham and O'Connell, 2012	**6yr, F, mandibular 1st molar**	Fissure seal on eruption, monitored radiographically for 5 years
	10yr, F , mandibular 2nd premolar	Monitored and restored on eruption, monitored for 6 years
	12yr, F , mandibular 2nd molar	Extraction
Kakade and Juneja, 2013	**7yr, F, mandibular 2nd molar**	Oral antibiotics, surgical extraction, reviewed at 1 month post treatment showed uneventful healing of extraoral sinus tract, patient referred to a plastic surgeon
Czarnecki et al, 2013	**4yr 3mo, F, mandibular 1st molar**	Surgical exposure and occlusal surface sealing with high viscosity GIC, reviewed at 44 months post treatment showed no sign of lesion progression
Ari, 2014	**12yr, M, mandibular 2nd premolar**	Oral antibiotics, Root canal treatment and composite resin restoration, no subsequent recall
Wong and Khan 2014	**12yr, F, mandibular 2nd molar**	Surgical extraction, no subsequent recall

is unknown as radiographs of unerupted primary teeth are seldom taken, with only a single case of PEIR has been reported in a primary tooth.[16]

Aetiology

The aetiology and pathogenesis of PEIR lesions is unclear and a number of theories have been proposed in the literature over the years. The original theory proposed by Muhler (1957) suggested that periapical inflammation associated with a primary tooth may have the potential to cause disruption of the protective dental epithelium of the permanent successor which would allow increased vascularisation and an influx of inflammatory resorptive cells that subsequently induce a resorptive defect on the crown of the developing permanent tooth.[17] However, the majority of the teeth affected do not have a primary predecessor. It has been suggested that the lesions may be developmental as certain sections of the tooth fail to mineralise fully leaving a dentine defect. This has been disputed by the fact that most lesions are progressive and by a study showing that the PEIR lesions become evident after completion of full crown development.16 Another commonly held theory in the past is that the lesions are cause by dental (unerupted) caries. The potential for bacterial invasion into dentine of an unerupted tooth, which is encased in its follicle, is unlikely; although two cases of histologically confirmed caries in unerupted third and second molars communicating with the oral cavity via a soft tissue defect, have been reported in the literature.[6,18]

Currently, the most accepted theory based on evidence from multiple case reports and histological studies is that PEIR lesions are due to resorption processes.[14,16] Seow et al (1996) hypothesised that resorptive cells possibly originating from the dentinal follicle or the surrounding bone enter the dentine though a break in the enamel surface (such as hypoplastic pits, surface cracks or lamellae). The mechanism of initiation of this process is unclear although there has been reported association of the lesions with ectopic eruption,[10,11] leading to suggestion of 'local pressure' as the initiating mechanism.[16] The main opposition to this theory is the reported non-progressive nature of some lesions.[19] Other authors have commented that rough or hypoplastic enamel may play a part in initiating the resorptive process, similar to that reported in cases of hypoplastic amelogeneisis imperfecta where the entire tooth crown (including enamel) is often resorbed.[20] More research is required in this area.

Histological presentation

In PEIR lesions, histological examination consistently show fibrovascular tissue with a low grade inflammation filling the resorption defect.[21,22] Peripherally there are mono or multinuclear cells in typical resorptive lacunae demonstrating some evidence of hard tissue repair with unstructured hard tissue resembling bone abutting resorbed dentine at reversal lines.[16] The origins of these resorptive cells and their pathways of entry as well as the mechanism of initiation of resorption are still unclear.

Clinical exploration of PEIR lesions prior to tooth eruption or bacterial invasion often reveals pink soft tissue within the resorbed dentine. However, the defect may be indistinguishable from dental caries if the lesion is explored after tooth eruption and bacterial invasion.[14] Histological reports of extracted teeth often show a retained integrity of the dentine layer immediately adjacent to the pulp, and the pulp showing no obvious inflammatory or reactive response even in cases where the PEIR lesion appears large and aggressive.[21-23]

Pre-eruptive progression of lesion

The literature reports various rates of progression of PEIR lesions; although most cases show slow progression until the tooth erupts with some lesions undergoing period of high activity.[3] Moskovitz (2004) reported a case of PEIR in a first permanent mandibular molar that did not progress radiographically for seven years.[19] Conversely, other authors have reported a rapid rate of progression of defects within the period of 6-12months,[14] and particularly in a case of amelogenesis imperfecta.[20] Hata et al (2007) also reported that the rate of progression can vary with different teeth in a single individual.[14]

Post-eruptive progression of lesion

Once the tooth breaks through the mucosa of the oral cavity, bacteria often enters into the resorptive lesion either through the 'original' enamel defect (through which the resorptive cells presumably entered) or via the natural porosity of the enamel.

A large carious lesion often results due to the bacterial ingress and subsequent further breakdown of dentine.[24] In some affected teeth, the pulp is encroached soon after eruption, so that a dental abscess quickly develops. Most authors have reported absence of symptoms until the lesion reaches the pulp and infection of the pulp occurred. The disproportionate size of the cavity compared to the time since the tooth has been erupted may be a diagnostic feature of PEIR.[3]

Hata et al (2007) have described a post eruptive, non-carious progression of PEIR in a permanent mandibular lateral incisor.[14] It is usually assumed that upon eruption the vascular supply to the resorptive lesion is cut off therefore, arresting the resorptive process. Hata, however, hypothesises that if the PEIR is communicating with the external tissue near the cementoenamel junction, the vascular supply may be maintained and the progression of the lesion will not arrest even after eruption.[14]

PEIR and "Occult caries"

Occlusal caries in fluoridated areas is said to account for 80% of new lesions.[27] However, due to the complex nature of occlusal anatomy and the pattern of spread, occlusal caries detection can be challenging. Literature has termed lesions that are clinically undetectable but discovered on radiographs as "occult"[28, 29] or "hidden"[30-32] caries.

Seow (2000) has proposed that a significant proportion of lesions diagnosed as occult caries may have originated from PEIR.[2] This is based on multiple reports of occult caries with evidence of radiolucent lesions prior to eruption.[10] The contribution of PEIR to overall prevalence of occult caries is unknown, however, one study demonstrates that half of the children that were diagnosed with occult caries at a school clinic, had radiographic evidence of PEIR.[11] This prevalence is likely to be higher if all children had radiographs taken during the pre-eruptive stage of teeth development.

"Fluoride Bomb / Fluoride syndrome"

It has been a popular belief that occult caries was caused by the widespread use of fluoride, with many occult lesions being termed 'fluoride bombs" or "fluoride syndrome".[28, 29] It was hypothesised that fluoride encourages remineralisation and slows the progression of caries in the fissure/pit enamel, while the carious process continues in the dentine, with the relatively intact enamel surface masking the lesion underneath.[33, 34] This hypothesis has been disputed by a number of studies.

One study based in Nederlands examined rates of occult caries in two cities one of which had optimal water fluoridation and the other which had low fluoride.[35] The study found that the rate of occult caries was 31% lower in the fluoridated town compared to the town that had low fluoride. Another, more recently published retrospective study examined the clinical radiographic data of 500 school children in Brazil in 1975 and 1996 for prevalence of hidden caries in first permanent molars. The study showed that in 1975 when there was no access to optimally water fluoridation and limited access to fluoridated dentifrices the prevalence of hidden caries was 51% higher, compared to 1996 when the reverse was true.[36]

Management

Seow et al (1999) suggested that a number of factors should be considered prior to treating a PEIR lesion including the size of lesion, the time of detection, the anticipated eruption time of the affected tooth, the likely progressive nature of the defect, patient compliance, caries risk, malocclusion and crowding.[11] Early detection and diagnosis of the PEIR lesion is important as it is more likely to provide a wider array of treatment strategies for a successful outcome.[5] Therefore, as PEIR is almost always an incidental finding on routine dental x-rays it has been recommended that all dental x-rays be examined and scrutinized routinely for PEIR.[3]

The principal objective of management is the removal of the resorptive tissue and restoration of the defect with an appropriate dental material.[3,8,11,19] If the lesion is small or slowly progressing or the tooth is soon to erupt than the management may involve conservative observation with periodic radiographic exposure until the tooth has erupted sufficiently to explore the defect, remove the contents and appropriately restore the tooth.[5,14,15,37,38] If however, the lesion is large and encroaching the pulp or deemed to be aggressive than the tooth may be surgically exposed and appropriately restored and the soft tissue (if recoverable) sent for histological investigation.[2,7,8,11,15,37,39-41] In some cases where the tooth destruction is so extensive that the pulp is involved and the tooth cannot be predictably restored the only option may be surgical extraction.[6, 8, 12, 20, 23, 38, 40] The prognosis of teeth affected by PEIR will depend on the size of the lesion and presence of pulpal involvement. However, the reported cases in literature even with small pulpal exposures generally show favourable outcomes after routine treatment, with reported absence of symptoms, maintenance of pulp vitality and continuation of root development.[14] Recurrence of resorption after curettage and restoration of the lesion has not been reported.

Case Report

A 5 year old healthy boy was referred for management of severe early childhood caries under general anaesthetic at the Logan Hospital, Queensland, Australia. The patient had a history of pain and

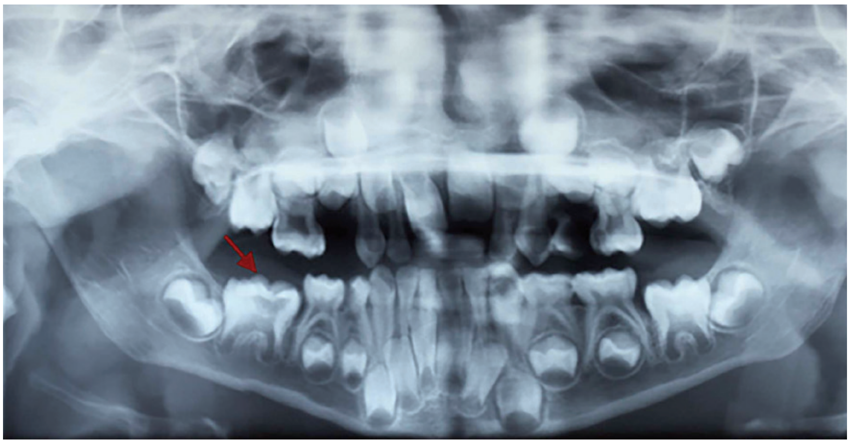


Figure 1. *Preoperative orthopantamogram*

Teeth present and erupted – 55, 53, 52, 62, 63, 64, 65, 75, 74, 73, 72, 71, 81, 82, 83, 84, 85. Teeth present and unerupted 17, 16, 15, 14, 13, 12, 11, 21, 22, 23, 24, 25, 26, 27, 37, 36, 35, 34, 33, 32, 31, 41, 42, 43, 44, 45, 46, 47. 18, 28, 38 and 48 are not visible. Caries is present on teeth 55, 53, 63, 64, 65, 74, 73, 83, 84, 85. 46 is unerupted an has a large radiolucency present in the dentine in close relation to the pulp. Temperomandibular joints are not adequately visualised. The bony texture is normal.

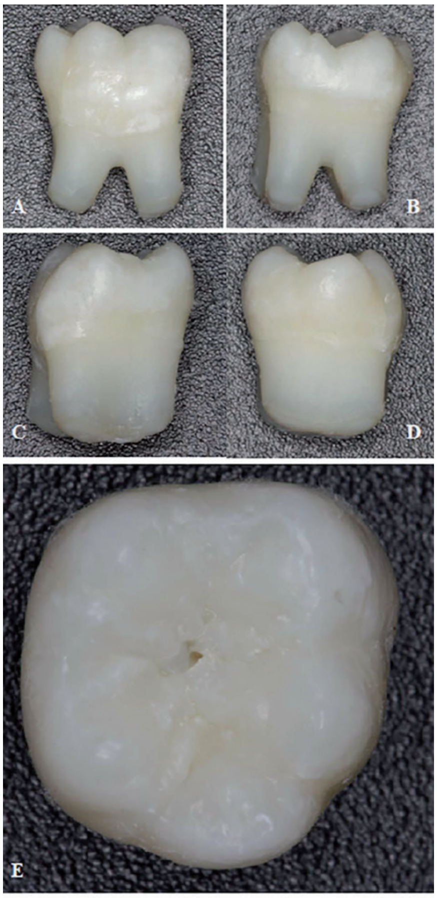


Figure 2. Images of fist permanent molar (46) after extraction
A – buccal view; B – lingual view; C – mesial view; D – distal view.
A, B, C & D all show normal morphology and incomplete root development coincident with the patient's age
E – occlusal view showing clear enamel defect in the occlusal fissure

swelling associated with grossly carious maxillary primary first molars and mandibular primary first right molar. The maxillary primary right first molar was extracted approximately 12 months earlier under local anaesthetic.
The patient's medical history was unremarkable. He was not taking any medications and had no allergies. The patient was born at full term by natural birth after an uncomplicated gestation. The patient's weight and length at birth and at presentation to the pre-operative examination were normal (50th percentile). He reached developmental milestones normally. The facial appearance as well as the hair and nails were normal. His profile was visually mesofacial.
Intraoral examination revealed poor oral hygiene and visible plaque deposits. Caries was present in all of the primary molars, primary canines as well as the maxillary primary left lateral incisor. An orthopanthograpm (OPG) exposed at pre-operative assessment confirmed the presence of caries in the primary teeth and revealed a large radiolucent lesion involving the dentine and encroaching the pulp of the unerupted mandibular permanent first right molar (46) (Figure 1). The 46 tooth was diagnosed with PIER based on the radiographic appearance. A number of treatment options, including their advantages and disadvantages, were discussed with the parent and patient regarding the unerupted 46 tooth. The options included monitoring and restoration after eruption, surgical exposure and restoration as well as surgical extraction. Due to the size of the lesion and the likelihood of long term endodontic and restorative complications, as well as the patient's high caries risk, a decision was made to surgically extract the tooth.
The patient was treated a week later under general anaesthetic as a day surgery case without any complications. The maxillary primary first left molar, the mandibular primary first right molar and the mandibular primary canines were extracted, the maxillary primary second left molar, the maxillary primary first left molar and the mandibular primary second right molar have been restored with stainless steal crowns, and the maxillary primary second right molar, maxillary primary canines, maxillary primary left lateral incisor and maxillary primary second left molar were restored with resin modified glass ionomer cement. The tooth 46 was surgically extracted via a distal relieving incision, simple elevation and closure using absorbable sutures. The patient was reviewed two weeks post-operatively. He recovered well and continued to attend six monthly preventative visits.

Gross appearance of 46 tooth

The tooth was inspected and overall morphology appeared normal with incomplete root development in line with the age of the patient (Figure 2). A clear enamel defect in the occlusal surface of the tooth was visible, while all other surfaces of the tooth appeared normal. The tooth was stored in a 10% formalin solution.

Cone beam computed tomography (CBCT) examination of 46 tooth

The tooth was imaged (Figure 3) using an Orthophos XG 3D Cone Beam CT Scanner (Sirona, New York, USA). The scan parameters were 8x8cm field of view, 160 micron resolution, 85kV and 5mA. The extracted tooth was mounted in wax, in the focal trough on a chin rest. The DICOM data set was analysed using Galaxis (v1.9.4 ID7) software (Sirona, New York, USA). Morphologically, the 46 demonstrated normal external crown and root formation (half root completion) consistent with the age of the patient.[42]
There was a prominent resorptive defect in the 46 crown with significant thinning of occlusal enamel intra-coronally. There was a focal defect in enamel of the occlusal surface centrally within the 46 occlusal fissure. The resorptive defect extended very close to the mesio-buccal, mesio-lingual and disto-lingual pulp horns but there was no definite evidence of destruction of the roof of the pulp chamber and extension of the resorptive defect into the pulp. No reparative dentine was evident. The resorptive defect extended into the lingual cusp tips and to the mesial dento-enamel junction. The resorptive defect also extended into the mid-buccal cusp tip.

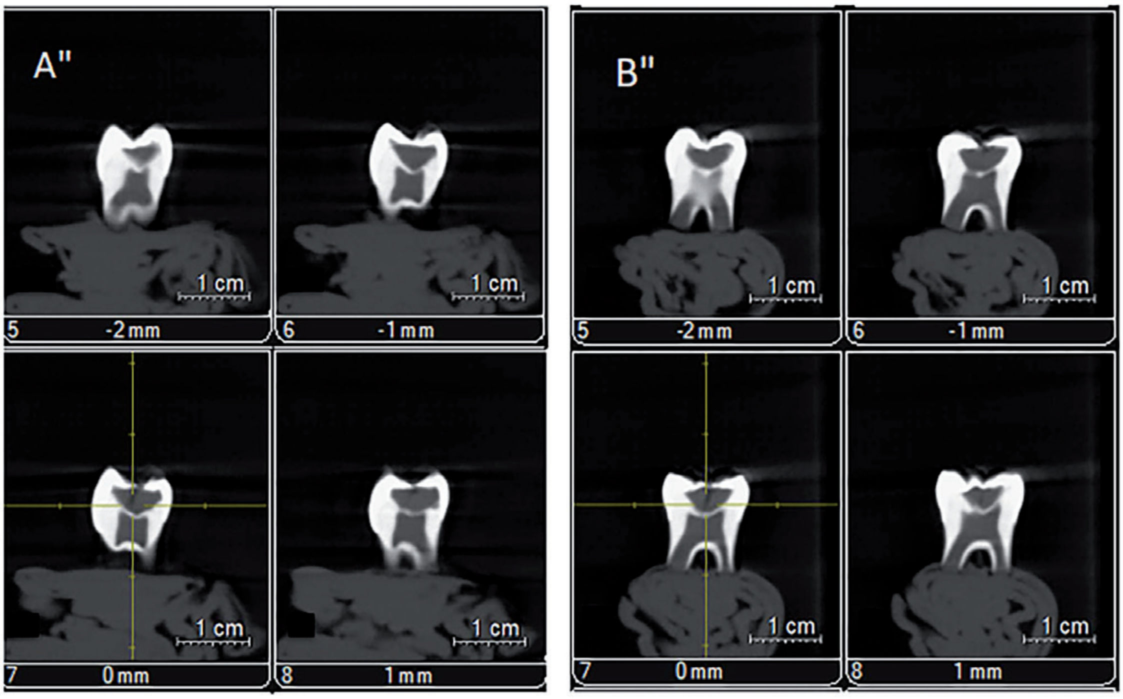
Examination of sectioned 46 tooth

The tooth was later sectioned exposing a fibrous tissue extending through most of the dentine with a very close association with the pulp (Figure 4). There was no evidence of reparative dentine formation. These findings were consistent with the radiographic findings.

Discussion

To the authors' knowledge, this is the first report of PEIR using cone beam computed tomography. This technique has the advantages of showing dimensionally accurate images of the intact tooth in all three planes compared to conventional two-dimensional radiography. Although, in the present case, the CBCT was used after the tooth was extracted and the three dimensional radiographic findings have not altered the original treatment rationale, the authors propose that the use of CBCT for PEIR lesions which are medium to large in size would add valuable insight of the exact extension, location and pulpal involvement of the lesion. This would aid endodontic treatment planning and in determining the surgical approach (occlusal or buccal/lingual).
This case report is also the first one to verify using CBCT, a definitive communication in the occlusal surface of the enamel with a PEIR lesion (Figs 3-5), which is the likely entry point for the resorptive cells into the dentine. In previous publications, enamel defects which have been associated with

Figure 3. Cone beam CT of fist permanent molar (46) after extraction
A – coronal (posterior to anterior) view; **B –** sagittal (buccal to lingual view)
The 46 has normal crown and root morphology consistent with the age of the patient. There is an extensive resorptive defect in the dentine of the 46 crown with a focal enamel defect in the occlusal surface. The resorptive defect extends very close to the pulp but no reparative dentine is evident.



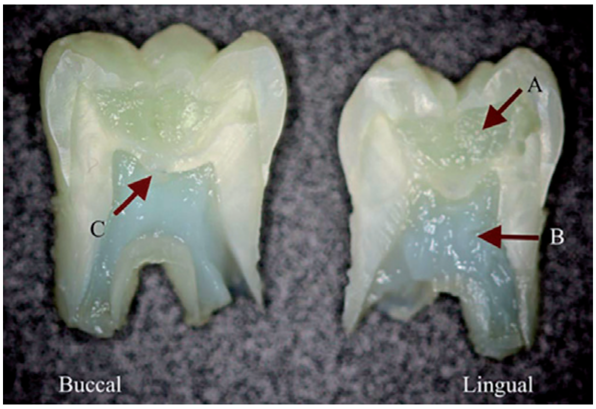


Figure 4 – Image of mesio-distally sectioned first permanent molar (46)
A – fibrous tissue occupying the resorption defect
B – pulp tissue
C – a thin layer of dentine remaining between the resorptive defect and the pulp
No inflammatory/ protective dentine formation is visible

PEIR lesions have been mainly reported on the buccal and lingual surfaces in the area of the cemento-enamel junction and these have been hypothesised to be the entry point for the resorptive cells.8 Others have commented that rough or hypoplastic enamel may be play a part in initiating the resorptive process.[20] On the other hand, it has been hypothesised that 'local pressure' may be associated with the initiation mechanism of the resorption, as PEIR occurs more commonly adjacent to or within ectopically positioned teeth.[10, 11] Variable rates of progression of PEIR lesions have been previously reported, although majority of cases show slow progression prior to tooth eruption,[3,19] and over half of previously reported lesions have extended less than one third of the width of the dentine.[8,12] Our case demonstrates an extensive resorptive lesion closely associated with the pulp as seen both visually and radiographically (Figs 4 and 5), which is less commonly reported. The use of cone beam computed tomography, provides an accurate, three dimensional representation of the extent of the PEIR and therefore can be a valuable tool in treatment planning extensive lesions.

The treatment options commonly described for PEIR range from conservative monitoring of the tooth to surgical extraction depending on the size and progression of the lesion, patient's age and caries rate.[7,11,19] There have been a number of papers that have reported successful management of large PEIR lesions that encroach the pulp by surgical exposure, curettage and restoration resulting in continual root development and maintenance of the pulp vitality.[37, 43] However, due to our patient's high caries rate an extraction was deemed most appropriate as this option would not predispose the child to potentially complicated and expensive restorative treatment in the future. This treatment rationale is supported by a number of published reports.[6,8,12,20,23,38,40]

As orthodontic problems may arise from early extraction of the first permanent molars, such as mesial tipping of the second permanent molar; orthodontic treatment may be required as part of the management.

Although relatively few case reports of PEIR have been published in the literature, the studies reporting on the prevalence indicate that the lesions are not uncommon.[4, 10-13] Furthermore, as PEIR is almost always an incidental finding on routine dental x-rays, all dental practitioners should be aware of their presentation and be vigilant when reading dental x-rays.[3] Early detection and diagnosis of the PEIR lesion is important as it is more likely to provide a wider array of successful treatment strategies.[5]

In conclusion, the present study shows that cone beam computed tomography aids in accurately determining the pulpal extent and the location of PEIR lesions for better restorative, endodontic and surgical management.

Why is this paper important to paediatric dentists?

- This paper provides a current update of PEIR with respect to aetiology, prevalence, diagnosis and management

• This paper shows that CBCT can be a useful diagnostic tool for determining the extent of the PEIR lesion and their pulpal involvement for improved management and treatment planning

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Not just baby teeth!

BEHIND THE SCENES FOR TREATING KIDS

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20TH BIENNIAL CONGRESS
ANZSPD 2020
Hotel Grand Chancellor
Hobart | 6-7 March 2020

International Keynote SPEAKERS

DR GREG PSALTIS

Dr. Greg Psaltis has been a paediatric dentist for 45 years. While most of his experience has been in private practice in Olympia, Washington, he served three years in the US Navy and now spends much of his time on two volunteer clinics in Mexico that he developed himself. His varied career has included authoring several articles for professional journals, speaking at every major dental meeting in the United States as well as several international meetings that have taken him to Germany, Mexico, Russia, Canada and Kuwait. His engaging style and sense of humor make learning both fun and interesting. Among his honors is the Gordon Christensen Speaker Recognition Award for excellence in dental education.

MARY ELLEN PSALTIS

Mary Ellen's professional writing on food and lifestyles has spanned over thirty years. Her interactions with chefs, farmers, and restaurateurs led to greater involvement in nutrition education and teaching. Mary Ellen speaks nationally on creating optimal nutrition and making thoughtful life choices. Her local classes focus on supporting people on their healthful life journeys. Mary Ellen's special interest is in aging well. Her mother, who lives nearby, is 93 and has a husband who recently celebrated his 99th birthday. Her dental assisting skills continue to improve on the frequent Mexican mission trips with her husband Greg.

Event HIGHLIGHTS

Behaviour management
- Paediatric pearls
- Anxiety

Nutrition
- Sugar bugs
- Foods and Fads

Leadership

Self care

TPHE – THE production house EVENTS

For any queries, please contact the Conference Secretariat The Production House Events:
Anna Cornwell - acornwell@tphe.com.au

2019 Professor Louise Brearley Messer
Post-Graduate and Under-Graduate
Essay Competitions

The Australian & New Zealand Society of Paediatric Dentistry (Inc) is once again holding its Annual Essay Competitions.

Post-graduate competition

The post-graduate competition is open to all Paediatric Dentistry post-graduate students who are currently enrolled in a Doctorate, Masters or a Post Graduate Clinical Diploma in a Paediatric Dentistry program at an Australian or New Zealand Dental School.

The topic for 2019 is:

We are into a new era in which parental-based research from internet searches and social media exposure is having a significant influence on what oral health care services they are wanting their children to receive. Discuss examples of services that may be requested by parents from you based on their research that may be deemed alternative to conventional oral health care practice. How you would approach each of these situations? Include in your answer evidence for and against both alternative and conventional treatment option outcomes.

A first prize of AU$2,500.00 will be awarded to the best entry.

Under-graduate competition

The under-graduate competition is open to all dental under-graduates or students enrolled in a graduate program leading to the student's first dental degree and who are enrolled in an Australian or New Zealand Dental School.

The topic for 2019 is:

Biosilicates have been found to have multiple applications in paediatric dentistry. What are they and are they as good as the alternatives?

A first prize of AU$1,500.00 will be awarded to the best entry.

The Essay should not exceed 3000 words. Bibliographic style should follow that of the Australian Dental Journal. Essays are to be the work of individuals only.

The deadline for the submission of Essays is Friday 25th October 2019

Entries should be submitted to the course co-ordinator who will select the "Best Entry" and email it to the Federal Secretary ANZSPD(Inc.), Dr C Lloyd at: federal.secretary@anzspd.org.au

The winner will be announced by January 10, 2020

RECEIPT OF YOUR ENTRY WILL BE ACKNOWLEDGED BY RETURN EMAIL.
If a receipt email is not received, please send a further email to fed.secretary@anzspd and /or carmell@westnet.com.au

See https://www.anzspd.org.au/viewStory/competitions-awards for conditions and notes on the award.

The ANZSPD Alistair Devlin Memorial Grant

Applications are now open for the 2019 Grant!

FULL members of ANZSPD **are now invited to submit applications** for the ANZSPD Alistair Devlin Memorial Grant.

At the ANZSPD Federal Council meeting in Melbourne in February 2014, it was decided that the existing ANZSPD Grant be renamed **The ANZSPD Alistair Devlin Memorial Grant in honour of Alistair's memory** and to acknowledge his most significant contribution to the society.

One grant per year will be provided to the value of AUD $2000 with eligibility restricted to current Full Members of ANZSPD (Inc.)

The grant is available for:

- **An oral health initiative** in Australia or New Zealand which may be an educational resource or a broad community initiative
- **A community research project** directly related to child oral health
- **Support for an oral health project** in Asia, Oceania or the Pacific which might be for materials, instruments, books for a school, etc.

Applications are open for the ANZSPD Alistair Devlin Memorial Grant on 1st June 2019 and **close on the 30th June 2019.**

Applications will need to contact the Federal Secretary ANZSPD (Inc.) (fed.secretary@anzspd.org.au) for full application guidelines.

Federal Council will then adjudicate.

The successful applicant will be required to provide a report to the Federal Council, suitable for publication in the Society's newsletter, Synopses, by September 2020.

The Federal Council may choose not to award a grant in the event of there being no suitable applications. **For more information** see the Competitions and Awards page on our website.

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Please do not contact Colgate for address correction.

Submissions

All text for inclusion in Synopses must be submitted to the editor by email.
Address email to steven@kazoullis.com
Please include your contact details with all submissions.

UP COMING EVENTS

1-4 May 2019
38th Australian Dental Congress
Adelaide Convention Centre
adacongress.com.au

23-26 May 2019
AAPD 72nd Annual Session
Chicago, Illinois, USA
annual.aapd.org

1-30 June 2019
Alistair Devlin Memorial Grant entries open
bit.ly/2TwRQPf

25 October 2019
Entries close for Louise Brearley Messer ANZSPD Undergraduate Essay Competition
Louise Brearley Messer ANZSPD Postgraduate Essay Competition
bit.ly/2U9xf8a
bit.ly/2CBPGIS

17-20 June 2020
IADT 21st World Congress on Dental Traumatology
Lisbon, Portugal
www.iadt-dentaltrauma.org

3-7 July 2019
27th IAPD Cancun Congress
Cancun, Mexico
www.iapd2019.org

5 December 2019
Abstracts due for ANZSPD-Colgate Research Award
bit.ly/2CCBGhT

6-7 March 2020
ANZSPD 20th Biennial Congress
Hotel Grand Chancellor, Hobart
anzspd2020.com.au

7-12 June 2021
28th IAPD Maastricht Congress
Maastricht, The Netherlands
www.iapd2021.org